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VIEWPOINT

Value of transoesophageal echocardiography before DC cardioversion in patients with atrial fibrillation: assessment of embolic risk

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Is it sound clinical practice to perform transoesophageal echocardiography (TOE) before direct current (DC) cardioversion or merely a "high tech" intervention? Let us review some facts about atrial fibrillation, including complications and treatment options.

Atrial fibrillation

Atrial fibrillation is the most common cardiac rhythm disturbance and although it is not usually life threatening, it is associated with important medical problems. The prevalence of atrial fibrillation is around 2% in the general population[1] and it affects 5% of those over 60.[2,3] The most feared complications associated with atrial fibrillation are peripheral embolisation and stroke. Loss of organised atrial contraction results in a decrease in cardiac output and may have considerable haemodynamic consequences in patients with cardiac hypertrophy, in those with poor systolic function, and in patients with valvar heart disease. Atrial fibrillation leads to the loss of the normal graded physiological chronotropic response to exercise.

The mean annual risk of stroke in chronic atrial fibrillation is 5% in untreated populations; however, the embolic risk varies in different populations. The risk is higher than 5% in patients with atrial fibrillation associated with valvar heart disease not treated with anticoagulation.[1] The mean annual risk of stroke in patients with non-valvar heart disease is 5%.[4] However, in certain subgroups of patients with non-valvar heart disease, such as those with coexisting hypertension, recent congestive heart failure, or a prior history of arterial thromboembolism the annual risk of stroke is > 7%.[4] Patients with lone atrial fibrillation, on the other hand, have a very low risk of embolisation (0·5%), particularly those patients who are less than 60 years old.[5]

Management of atrial fibrillation includes restoration of sinus rhythm, treatment with antiarrhythmic drugs, and anticoagulation. Conversion to sinus rhythm occurs after DC cardioversion or spontaneously after initiation of antiarrhythmic therapy. In patients with atrial fibrillation of less than 2–3 days' duration the conversion rate was high but this rate fell significantly when atrial fibrillation had been present for longer.[6] Heart rate control alone may be an alternative treatment.

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There have been several prospective trials to compare the efficacy of warfarin, aspirin, and placebo in stroke prevention in non-valvar atrial fibrillation.[7–10] Recently, pooled data from five randomised trials showed a 68% reduction of strokes in patients treated with warfarin and a 36% reduction in those treated with aspirin.[11] Low-dose warfarin has gained increasing attention as a possible treatment option to reduce the incidence of intracranial bleeding in patients treated with warfarin.[12] The risk of intracranial haemorrhage is 0·8–2·5% per year.[7–10] Although the reduction in the rate of embolic strokes is much better with warfarin, certain groups—that is, patients with a low risk of stroke or a high risk of an intracranial bleed (patients over the age of 75 years)—may be candidates for aspirin. Further studies are needed to establish the relative effectiveness of aspirin.

DC Cardioversion

If pharmacological conversion to sinus rhythm fails, subsequent DC electrical cardioversion is initially successful in about 90% of patients.[13] Maintenance of sinus rhythm is variable and depends on several factors including the duration of atrial fibrillation. The number of patients remaining in sinus rhythm after 6–12 months may be less than 50%.[14]

DC cardioversion carries its own risk of peripheral embolisation, which is associated with the duration of atrial fibrillation before cardioversion, atrial appendage function before cardioversion, and other factors. The mechanism of embolisation during cardioversion has been thought to be due to dislodgement of pre-existing clot in the atrium or atrial appendage. Recent studies using transoesophageal echocardiography suggest that such peripheral embolisation also may be due to de novo formation of thrombi after cardioversion.[15–16] Successful cardioversion is associated with a 5–7% incidence of embolism in patients who have not been treated with anticoagulants.[17,18] The incidence of strokes was reduced to less than 1·6% in patients "pretreated" with anticoagulation.[19–21] The American College of Chest Physicians (ACCP) recommends that patients undergoing cardioversion of atrial fibrillation that has lasted for more than two

days should be given anticoagulants for three weeks before cardioversion.[22] The practice of foregoing anticoagulation if a patient has had atrial fibrillation for less than 48–72 hours is based on clinical experience and a published report.[21]

After successful DC cardioversion with return to sinus rhythm a risk of peripheral embolisation persists until atrial appendage function is restored. This may take several weeks or months.[23] Therefore, the current recommendation of the American College of Chest Physicians is to anticoagulate for four weeks after DC cardioversion.[22]

Role of TOE

What is the role of TOE in the treatment of patients considered as candidates for DC cardioversion? First, can TOE identify clots present in the left atrium or left atrial appendage before cardioversion? Second, is it safe to forego three weeks of anticoagulation before DC cardioversion if a TOE is negative for clot? Third, can TOE before cardioversion identify the risk factors for clot formation after cardioversion that might justify shortening or abolishing the post cardioversion anticoagulation? Fourth, is it cost effective to perform TOE before cardioversion?

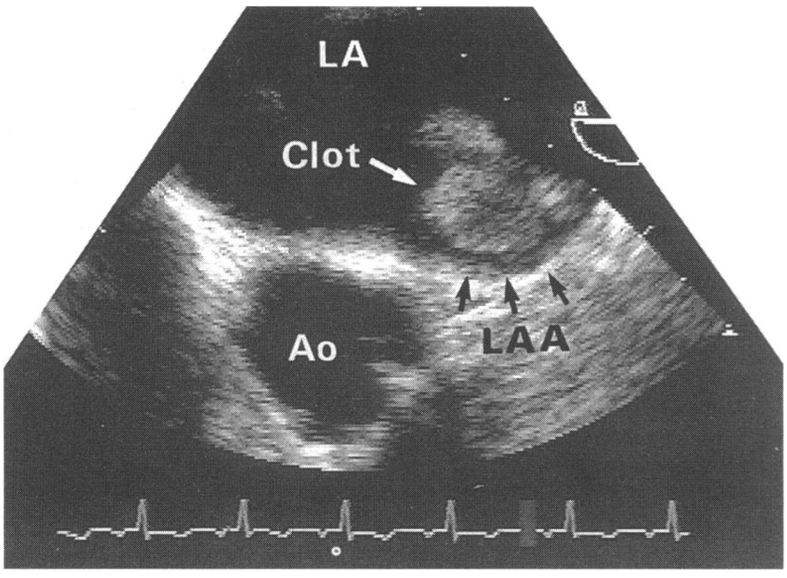

Figure 1 A large clot visualised at the entrance to the left atrial appendage (LAA). LA, left atrium; Ao, aorta. The left atrial appendage is slightly foreshortened in this image.

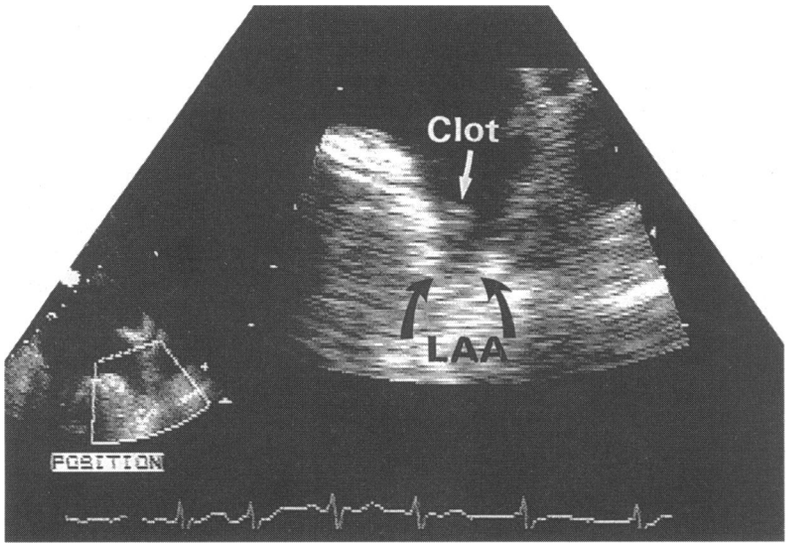

Figure 2 A small clot anchored on the medial wall of the left atrial appendage (LAA), near the tip of the appendage (black arrows). This central image was magnified from a scan plane of the left atrium and left ventricle (lower left corner).

IDENTIFICATION OF CLOT BEFORE CARDIOVERSION

Previous reports suggest that TOE is a highly sensitive way of detecting clots in the left atrium and left atrial appendage (figs 1 and 2).[24–26] However, the size of the smallest resolvable clot has not been systematically determined.

Patients treated with anticoagulation before cardioversion have not routinely undergone echocardiographic studies to rule out clot. It has been assumed that they are relatively "safe" from stroke associated with cardioversion—none the less, the stroke risk in this anticoagulated population is still about 1·6%. Also patients with atrial fibrillation for less than two days have been cardioverted without anticoagulation. Recent studies, however, suggest that both these groups of patients are at some risk of harbouring intra-atrial clots. Manning *et al* reported persistence of clot in one patient after five weeks anticoagulation.[27] In another study TOE in two patients showed persistence of intra-atrial clot despite three months of anticoagulation.[28] The belief that patients with atrial fibrillation of less than 48–72 hours' duration have no significant risk of stroke if they are cardioverted without anticoagulation was recently challenged by Stoddard *et al.*[29] They found a clot in the atrial appendage in 20 (14%) of 143 patients who had had atrial fibrillation for less than three days. This finding does not necessarily mean that 14% of patients would have had a stroke had they been cardioverted—because not all patients with an intracardiac clot will have an embolic event after cardioversion. Nevertheless, the findings of this study question the practice of DC cardioversion in patients who have had atrial fibrillation for less than three days when an intracardiac clot has not been excluded.

ABOLISHING PRECARDIOVERSION ANTICOAGULATION

Manning *et al* reported a study of 94 patients studied with TOE before cardioversion.[27] Fourteen atrial thrombi were identified in 12 patients (13%). Cardioversion was deferred in all 12 patients. Seventy eight of the 82 remaining patients without thrombi underwent successful cardioversion to sinus rhythm, all without long-term anticoagulation. None of these patients had an embolic event. Short-term anticoagulation with heparin, for a mean of 2·2 days, was given to 80 patients before cardioversion. Fatkin *et al* reported a study of 66 patients examined with TOE before cardioversion.[15] Left atrial

thrombus was detected in one patient (1·4%) and cardioversion was cancelled in this patient. Thromboembolic complications occurred in four patients (6%), none of whom had evidence of left atrial thrombus before cardioversion. The anticoagulation status of these patients varied: one patient received aspirin alone, one patient received subcutaneous heparin twice daily and two patients had subtherapeutic concentration of anticoagulant. This study shows that thromboembolic complications can occur in the absence of demonstrable clot if patients are not adequately anticoagulated at the time of cardioversion. Black *et al* reported on 40 patients examined with TOE before cardioversion.[16] These patients were not anticoagulated and had non-valvar atrial fibrillation. Five patients were found to have thrombi before cardioversion and cardioversion was therefore cancelled in all of them. Of the remaining 35 patients, 25 had successful cardioversion. A stroke occurred within one day after successful cardioversion in one patient. Repeat TOE showed a new thrombus in the left atrial appendage and an increase in left atrial spontaneous contrast. These studies raise the question of the mechanism of stroke. Small thrombi not identified by echocardiography may be present before cardioversion. Alternatively, thrombi may occur because of "acute atrial stunning", possibly induced by electrical cardioversion, that may predispose to blood stasis and thrombus formation after the procedure. These reports suggest that all patients should be treated with intravenous heparin during DC cardioversion. At the time of writing this report there are no published data to suggest that any patient had an embolic event when treated with short-term anticoagulation with intravenous heparin before cardioversion, if TOE was negative for clot before the procedure and the patient was anticoagulated for four weeks after cardioversion. However, the efficacy of such treatment needs to be examined in a larger study.

TOE AS A TOOL TO ASSESS PATIENTS AT RISK OF STROKE AFTER DC CARDIOVERSION

The function of the left atrial appendage may not return to normal immediately after restoration of sinus rhythm. Evidence of atrial function can be delayed as long as three months[23 27] and the left atrial appendage may recuperate at a different time. Because most clots are lodged in the left atrial appendage, the function of this part seems to be the most important.

Spontaneous contrast is one factor clearly associated with thrombus formation in the atrial appendage. This has been reported by several groups[30–32] and Fatkin *et al* found spontaneous increases in echo contrast after cardioversion in some patients, possibly due to "atrial stunning" as a result of DC cardioversion.[15] In patients with new or worse left atrial spontaneous contrast after cardioversion, atrial fibrillation had been present longer, the mean number of shocks was greater, and mean energy delivered was higher than in those with unchanged spontaneous contrast.[15] TOE before cardioversion did not prospectively predict which patient would have an increase in contrast.

TOE was used to study Doppler velocities of filling and emptying of the left atrial appendage as well as its ejection fraction. Healthy individuals had a quadriphasic pattern, consisting of two diastolic forward (emptying) flow waves, each followed by a backward (filling) flow wave.[33] Filling and emptying velocities of 0·48 m/s are normal.[34] Patients in atrial fibrillation have lower velocities than patients in sinus rhythm. No high risk group among those with atrial fibrillation has been identified; however, the contraction pattern and size of the left atrial appendage may be a risk factor in the formation of thrombus in the left atrial appendage. Patients with non-rheumatic atrial fibrillation had one of two patterns of Doppler velocities in the left atrial appendage[35]: either well defined filling and emptying > 0·25 m/s (high profile) or irregular low velocities of < 0·25 m/s (low profile). Of 29 patients studied, embolism occurred in five low profile patients and in only one high profile patient. Also 80% of low profile patients had spontaneous echo contrast but only 5% of patients with high profile showed such contrast.[35] Left atrial spontaneous contrast, which has been linked to the formation of clots in the left atrial appendage in several studies,[30–32] is more likely formed at lower velocities.

Further studies are needed to evaluate whether any measures of left atrial appendage function assessed before cardioversion are predictive of thrombus formation after cardioversion. Such indices include Doppler emptying and filling velocities, left appendage size, atrial appendage ejection fraction, and spontaneous echo contrast.

COST BENEFIT OF TOE BEFORE DC CARDIOVERSION

The cost associated with patients treated with conventional therapy (three weeks of anticoagulation before cardioversion and four weeks of anticoagulation after cardioversion) was recently compared[36] with the cost of treating patients by brief heparinisation after a negative TOE and followed by DC cardioversion and subsequent anticoagulation after cardioversion. If the incidence of stroke was reduced from 1·6% with conventional treatment to 0·9% with the proposed approach, the cost would be equal in both groups. In other words the cost to the individual and to society of only one stroke quickly justifies the cost of many transoesophageal echocardiograms. Not addressed in this study, but also important, is the added convenience of DC cardioversion performed during the initial hospital admission for atrial fibrillation.

Conclusion

- TOE seems to be an accurate method for detecting clots before cardioversion. This

technique may be helpful in all patients whatever their anticoagulation status.

- All patients should be anticoagulated at the time of cardioversion (intravenous heparin) even when the TOE is negative for clot.

- If TOE before cardioversion is negative for thrombus, it appears safe to forego three weeks of anticoagulation before cardioversion.

- Sustained anticoagulation should be given routinely for four weeks after successful cardioversion. It is hoped that future studies will establish whether TOE can reliably predict those at no risk of thrombosis after cardioversion who do not need anticoagulation after the procedure.

TOE before cardioversion has undoubtedly enhanced our knowledge of the possible mechanisms of embolisation, atrial function, and atrial appendage function. Whether or not a physician or institution uses TOE to evaluate patients with atrial fibrillation before cardioversion is a matter of local economics, priorities, and resources. Research is needed both to define more precisely groups at risk of embolisation after DC cardioversion and to provide guidelines for anticoagulation practices after cardioversion.

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